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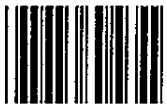

PRICE

COUNTY OF SAN DIEGO

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DECL.

JOHN J. SANSONE, County Counsel (State Bar No. 103060)
County of San Diego
DIANE BARDSLEY, Chief Deputy (State Bar No. 81525)
By RICKY R. SANCHEZ, Deputy (State Bar No. 107559)
1600 Pacific Highway, Room 355
San Diego, California 92101-2469
Telephone (619) 531-4874

Attorneys for Defendants County of San Diego, Jim Roache, John Groff, Steven Clause, Mark Tally and Samuel Sheppard

IN THE UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF CALIFORNIA

ANN PRICE, et al., Plaintiffs, v. COUNTY OF SAN DIEGO, et al., Defendants.	No. 94-1917-R(A.B.) TRIAL TESTIMONY DECLARATION OF DR. TOM NEUMAN, M.D.

I, TOM NEUMAN, M.D., declare:

I am a physician duly licensed to practice medicine in the United States. I am employed by the University of California San Diego Medical Center in the Emergency Department, as a Professor of Medicine and Surgery. I am Board Certified in Internal Medicine, Pulmonary Disease, Emergency Medicine and Occupational Medicine. My C.V. more thoroughly reviews my academic and professional credentials. I have previously testified as a medical expert in a court of law and in that capacity, I was retained by the Office of County Counsel. I have generated opinions regarding the hogtie restraint and its effect on respiration and ventilation, the accuracy of Dr. Donald Reay's work on positional asphyxia, and the cause of Daniel Price's death.

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(i) DR. REAY'S FLAWED HYPOTHESIS

The proposition that the prone hogtie restraint precipitates asphyxiation was advanced by Donald Reay, M.D., in a 1988 paper entitled "Effects of Positional Restraint on Oxygen Saturation and Heart Rate Following Exercise."

There are serious flaws with Dr. Reay's original paper. These flaws are serious enough to invalidate any conclusions drawn from that work, and therefore any diagnosis which relies upon the hypotheses set forth therein is therefore likewise flawed and medically unsound. The import to this case is that Daniel Price did not experience an asphyxial death by reason of the hogtie restraint. I have already discussed the many serious flaws of Dr. Reay's paper in my deposition and prior summary judgment declaration.

First, there are the flawed oxygen saturation measurements Dr. Reay reported. He found, and I quote, "drop in peripheral oxygen saturation during exercise and recovery, vary to levels of 85% to 90%." That is an extraordinarily abnormal response, as normals do not drop their oxygen saturation during exercise. As a normal person doesn't drop their oxygen saturation during exercise to 85%, one cannot logically ask what the clinical significance of such a drop in a normal after exercise would be. Hypothetically, if a normal individual's oxygen saturation levels were driven down to 85%,(by some other mechanism), that would represent a PO_2 somewhere between 50 and 65mmHg of mercury. Under those circumstances, a person almost certainly would be confused, profoundly short of breath, and major physiological alterations would take place. Hypothetically, this would be the equivalent of going to an altitude of somewhere around 15 thousand feet within minutes.

The erroneous oxygen saturation measurements reported in Dr. Reay's paper are attributable to the fact that the wrong type of device was used for making these measurements. Dr. Reay used a pulse oximeter, which numerous papers have shown can produce erroneous measurements of peripheral oxygen saturation during exercise. The drop in peripheral oxygen saturation during exercise which Dr. Reay reported is something that simply does not happen in normal people. As a result, either his subjects were markedly abnormal, or there was something wrong with his data collection. The latter is more likely because desaturation with exercise bespeaks very significant cardiopulmonary disease. Such a response is not that of an individual with slight clinical disease, but rather the response of an individual with severe pathology.

Another flaw in the paper is the logic employed. Even if Dr. Reay's data were accurate, which they are not, he can only conclude that after exercise and placement in the hogtie, his subjects experienced a prolongation in recovery time. Even if a recovery of O_2 saturation was necessary, just the mere prolongation of recovery time doesn't explain why somebody would die from this position. It only would suggest why it might take them a little bit longer to return to normal. In fact, the differences in recovery time he reports are only approximately half a minute. So the logic of why somebody, who takes half a minute longer to recover, should die escapes me entirely. If one graphs Dr. Reay's data, it is upon the termination of exercise that his subjects are at their worst. Whether restrained or nor, they only improve from that point onward. It is illogical to conclude they will die by asphyxia when they are "recovering." At any rate, Dr. Reay's observation that it takes slightly longer for O_2 saturation to

return to normal is absolutely incorrect because normals do not desaturate with exercise. Rather, their arterial PO_2 improves, therefore, there is no oxygen desaturation from which to recover.

Dr. Reay's paper is also flawed in the statistics he uses. He says that the mean time to recovery of oxygen saturation is 1.28 minutes during positional restraint and .95 minutes under the control circumstances. Dr. Reay presents the actual timed data in table number one of his paper. We, at the university, re-analyzed his data using a Student "t" test (the test Dr. Reay reportedly used) with our statistical package. A "t" test is a test that asserts the statistical significance of the difference in the means of groups and looks at the amount of deviation you have from the mean by each individual component that makes up the group. So, for example, the mean of the unrestrained group is .95 minutes to recovery. However, there is a range of .23 to 1.23 among the individuals of the group. What the "t" test does then, is it makes a computation based upon this variation of the groups. Then, it looks at the restrained group in the same manner. The "t" test then compares these groups to see if you can really say whether the difference between these groups occurred by chance alone. According to the numbers in Dr. Reay's table, the difference between the recovery times of the oxygen saturation in the restrained and unrestrained groups is so small that it must be considered to have happened by chance. The "P" value is equal to .126, which means there is at least a 13% chance that this happened randomly. A methodological research rule is that when something happens by chance more than 5% of the time, we reject it as being associated.

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Another technical flaw in his paper occurs where he refers to oxygen saturation and his placement of people in the hogtie position. He says, "Measurements were continued until base line oxygen saturation returned." But, base line oxygen saturation is never really defined. A normal person has an oxygen saturation somewhere in the neighborhood of 96% to 99%. If you start out at 98%, when you get to 96%, is that base line? If you start out at 96% and you get to 95%, is that base line? This cannot be determined from the paper. Furthermore, a pulse oximeter is simply not an appropriate instrument to measure small differences in oxygen saturation in the high ninety range. Because the oxygen saturation curve flattens out at higher saturations, a large change in PO_2 is required to have an effect on oxygen saturation at that level. So, absent PO_2 measurements, one has no idea what return to base line means.

Another important flaw in Dr. Reay's manuscript is the lack of direct measurements of pulmonary function. If one is going to hypothesize asphyxia occurred, one simply must measure ventilation to see if changes of a sufficient magnitude to cause asphyxia do really occur. By Dr. Reay's own definition at page 16 of his deposition, asphyxia requires hypoxia (decreased PO_2) and hypercapnia (elevated PCO_2) occur.

Thus the final major flaw is the lack of measurement of PCO_2. PCO_2 and alveolar ventilation are inversely related. Whenever ventilation goes down, PCO_2 goes up and vice-versa. Thus one must measure PCO_2 to functionally determine whether ventilation is reduced in a way which produces asphyxia. Obviously such measurements were not made by Dr. Reay.

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These flaws are important because they demonstrate that Dr. Reay's paper cannot be relied upon to support the hypothesis that the hogtie position precipitates an asphyxial death. Prior to our restraint study at UCSD, Dr. Reay's paper was for all intents and purposes, the only experimental evidence that existed which suggested people would have respiratory consequences when put in the hogtie position. Subsequent to Dr. Reay's paper, his paper was interpreted to mean that people died because of the hogtie position. Dr. Reay also advanced the proposition that people died because of the hogtie position. But he was not the only one. There were a series of case reports by authors relying on Dr. Reay's work which suggested that the deaths were due to the hogtie position. This then was a self-fulfilling prophesy based upon bad science where no one looked at the experiment in a critical fashion, but rather merely accepted it at face value.

(ii) UCSD STUDY

My training is in respiratory physiology. When I was first asked to look at Dr. Reay's paper, my immediate observation was that there was something dramatically wrong with the way this study was done because normal people don't desaturate with exercise. Also immediately apparent to me was the paper's logical flaw in deducing death resulted from prolonged recovery.

To scientifically examine the hypothesis that hogtying induces asphyxia, we, at UCSD Medical Center, did the study described in the paper entitled Custody Restraint Position and Positional Asphyxia. That paper has been accepted for publication in the Annals of Emergency Medicine and is scheduled to be published in the journal's November 1997 issue. An abstract of the paper was published in May

1997 in Academic Emergency Medicine, Vol. 4, No. 5. For our paper to be accepted for publication, it was peer reviewed by referees knowledgeable in the field who reviewed the manuscript to look for problems with scientific method, data collection, analysis and conclusions.

For example, I am the Editor in Chief of a journal and it is my responsibility to ultimately decide which manuscripts get published. The process is basically as follows: The editors receive a manuscript and then send it out to individuals who are familiar with the subject and methodology, and who are in a position to scientifically evaluate a manuscript as to its value. Typically there are a minimum of two reviewers, although in larger journals there may be more reviewers. These individuals look at the manuscript, review it carefully and then send back a whole series of comments about the manuscript that relate to the way its written, to its scientific method, to the statistics that are used, and to the conclusions that are drawn from the information. Those criticisms are then related back, in an anonymous fashion, to the author. The author then has an opportunity to revise the manuscript to meet the criticisms that have been made. The manuscript is then resubmitted to the editor of the journal and the editor of the journal can then either (seeing that all the criticisms have been answered) accept the manuscript, reject it, or send it back to the referees for another review. And then the process begins again until an ultimate decision is made as to whether a manuscript ought to be published or the manuscript is rejected. I do not believe that Dr. Reay's article was adequately peer reviewed. There are so many serious flaws in it that it was either not peer reviewed at all or it

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was reviewed by people who knew little to nothing about exercise physiology.

Basically, our study looked at gas exchange in the prone hogtie position, and pulmonary function in the same prone hogtie position. The people examined in the study included persons with Price's body habitus (shape, size, morphology, etc.) and body mass index.

It is critically important to look at gas exchange because that's the sine qua non of whether or not a particular activity affects respiratory function. If I were to ask you to breathe only through your nose, you would have no trouble walking up and down this corridor. On the other hand, you might have a great deal of trouble doing a mile sprint with your mouth closed. There would be interference with ventilation under these circumstances. So, the question then becomes, will we not only detect a change in pulmonary function with the hogtie position, but more importantly, will that change in pulmonary function have an effect upon oxygen levels and carbon dioxide levels in the blood. We expected to find an effect of position because prior literature demonstrates there is an effect from the supine position (on your back). Investigators had looked at the supine position in the past, and we know there is a difference between individuals seated upright and subjects being supine. As it turns out, no one had ever looked at the prone position before, and obviously no one had ever looked at the hogtie position. The question that had to be addressed was: Were these differences going to be of a magnitude that was going to be sufficient enough to interfere with an individual's oxygen and carbon dioxide levels in their blood and, if that did interfere, how much did it interfere? Thus, we conducted the experiment.

The experiment was conducted by examining subjects' pulmonary function before exercise, in the sitting, supine and prone positions. After that, subjects exercised on a bicycle ergometer at 175 watts. This represents a very strenuous level of exercise as evidenced by a mean heart rate of over 160 beats per minute in the subjects. Certainly this represents a much higher level of exercise than Dr. Reay employed in his work. As it turns out, the level of exercise used in our study was near maximal exertion for most subjects. After that exercise period, subjects sit and oxygen levels, carbon dioxide levels and pulmonary function tests were measured again. We waited for the subjects' heart rate to go below 100 beats per minute,and then they exercised again. At the end of that exercise period, we immediately put them in the hogtie position. We left them in the prone hogtie position for 15 minutes and during that period measured oxygen levels in their blood, carbon dioxide levels in their blood, and pulmonary function in that position. We then compared the results from these two time periods.

There are two major aspects to the study. Number one: what effects were there on gas exchange and number two: what effects were there on pulmonary function (the mechanical ability to move air in and out of the lungs). The short answer to the gas exchange question is - there is no effect. The single most important measurement to demonstrate that there is no effect on the person's ventilatory response to that degree of exercise, was that carbon dioxide levels in the blood were exactly the same in the two groups. That is critical because ventilation determines what your carbon dioxide level is. Ventilation does not necessarily determine what your oxygen level is, although, in most circumstances they are related. The relationship

between the degree of ventilation and the carbon dioxide level in your blood is very, very specific. Therefore, since the carbon dioxide level was the same in both of these groups, not only was there no effect, but there wasn't even an effect to suggest that the subjects breathed a little less than they would have liked, for that level of exercise. The hogtie group's ventilation was exactly and precisely as much as the people who were not hogtied. And that's critically important. Oxygen levels were of course, no different between the groups. Exercise improved the PO_2 in both time periods. This is to be expected because in exercise there is a tremendous improvement in the way the heart and lungs work together to supply oxygen to the body. Teleologically, this makes a great deal of sense. We have a tremendous reserve for doing more exercise. We do not need the heart and lungs to be terribly fine-tuned when we are just sitting, because there isn't that much demand on us. But when the lion is chasing us, we need to have everything working as well together as possible and that's exactly what happens when you start to exercise. Your heart and lungs work better together so that they become more efficient. The partial pressure of oxygen in your blood then goes up not down.

As far as our findings concerning pulmonary function, there is of course a decrement in pulmonary function with these changing positions. The pulmonary function referred to here are the mechanical volumes of gas that move in and out of your lungs. Pulmonary function tests measure how much air you can deliberately move in and out of your lungs and how fast you move the air in and out of your lungs. There is a difference between your maximal abilities in various positions. There was about a 7% reduction in the total amount of air that you can take in, in both the supine and prone

positions and 13% reduction in the prone hogtie restraint position. This is a highly statistically significant difference; meaning that the differences we identified here did not occur by chance. In other words, there was in fact a real difference, and this difference did not occur due to chance alone. We are not talking about how clinically important the differences were, we are merely talking about whether the differences occurred by chance or not by chance when we talk of statistical significance. In fact, there is no clinical significance to these changes. By that I mean, if you took the results of the pulmonary function tests from a subject and looked at them, you would interpret them as normal. In other words, the change that occurred to the individuals was small enough that it's still remained within the range of normal.

There are some vary well defined criteria that we use to make sure that the subjects' test results are reliable. The American Thoracic Society has set a criteria requiring three tests of every volume on a subject and those three tests have to be within 5% of one another. The American Thoracic Association's criteria were employed in our study.

The blood gas measurements of our study were done in several different ways. We used a pulse oximeter on the subjects' ear, we used a pulse oximeter on their finger, and we put in an arterial catheter and took blood samples. Blood samples were then divided into different sub-samples (aliquots). They went to two separate arterial blood gas machines and a third aliquot went to a co-oximeter. The pulse oximeter oxygen saturation measurements were done to see whether we could have the same bad luck with the pulse oximeters as Dr. Reay did when he recorded what had to be erroneous readings. Splitting the

allocated blood into three samples for two separate arterial blood gas machines and a third going to a co-oximeter is the standard for scientific accuracy. Not only are you getting a blood sample to directly measure PO_2 and PCO_2, but you are also testing one machine against another machine at the same time to make sure that the results are the same, and then the co-oximeter is the "gold standard" method to determine how much oxygen is actually in the blood. This is because when you put a sample into a blood-gas machine, you are really measuring the partial pressure of oxygen above the blood. When you use a co-oximeter, you drive all of the oxygen off of the blood and measure directly how much oxygen comes off. This is the methodology that's used in the best scientific laboratories to determine how much oxygen is in blood.

The conclusion of our study is really very straight forward. The ventilatory embarrassment secondary to the hogtie position does not cause any abnormalities in gas exchange, therefore, one cannot imply that abnormalities in gas exchange have caused somebody's death. You cannot say that a decreased level of oxygen due to the hogtie position caused somebody's death because the oxygen level doesn't go down in the hogtie position. You cannot say an increased level of carbon dioxide in the hogtie position caused somebody's death because the carbon dioxide level doesn't increase in the hogtie position. You cannot say alterations in Ph or other blood chemistry (related to exercise) in the hogtie position caused somebody's death because those are no different than in the non-hogtie position. In other words, from a respiratory or gas exchange exercise point of view, putting somebody in the hogtie position has no effect as far as their gas exchange is concerned. Therefore, it cannot kill by asphyxia.

I'm not the only one who states that desaturation does not occur with exercise, please look in any textbook of respiratory or exercise physiology. PO_2 goes up with exercise, therefore the notion that in the hogtie position it takes longer for the PO_2 to return to normal, is simply wrong. Our study concurs with the physiology that's been published and established for years. It is only Dr. Reay's study that is diametrically opposed to the material in standard tests of exercise physiology. Why it has gained such popularity is inexplicable.

(iii) OPINION RE: DEATH

After reviewing the materials in this particular case, including the analysis of Dr. Reay's paper, our study, the autopsy report, the various depositions of Dr. Reay, Dr. Eisele, the deputies and witnesses, Daniel Price's medical and psychological records, I am convinced that Mr. Price did not asphyxiate due to the hogtie position. Rather, the most obvious cause of death is toxic delirium secondary to methamphetamine abuse, which in turn caused Mr. Price to experience a cardiac arrest.

Toxic delirium is a syndrome, a whole constellation of signs and symptoms that are seen in people who use methamphetamines. One aspect of the syndrome is delirium. Methamphetamine also makes you psychotic, which means the person can't test reality. From records I reviewed, Mr. Price had a chronic problem with methamphetamine abuse and his course is typical of the progression seen in regard to mental status associated with methamphetamine. In addition, he had abnormalities in his heart, which are consistent with chronic methamphetamine abuse, he had a very high temperature, and this tremendously high temperature is also typical of toxic delirium due to methamphetamine. He went on to develop rhabdomyolysis, which is

typical of methamphetamine toxic delirium as well. Rhabdomyolysis is the breakdown and death of muscle cells with the release into the bloodstream of the enzymes that are normally within the muscle cells, CPK being the most commonly measured, and that was elevated in Mr. Price. Rhabdomyolysis in turn can cause renal failure, and that happened to Mr. Price as well. So, there is a very typical picture of a person with a toxic delirium secondary to methamphetamine abuse. The concept that his blood levels of methamphetamine were not high enough to cause his death is one that I simply disagree with the pathologist about. It has been clearly established that there is a very poor relationship between blood levels of amphetamines and whether or not you get into medical trouble from them. That the post mortem methamphetamine blood level was .03 micrograms per milliliter is not particularly revealing of how much Price had in him at the time of the incident because people will completely clear methamphetamine from their bloodstream in 48 to 72 hours. Reading from Goldfrank's book Toxicologic Emergencies, the section entitled What Are The Clinical Effects of Amphetamines we see that Mr. Price exhibited many of the clinical symptoms of methamphetamine toxicity: "The clinical effects of amphetamines are related to the stimulation of central and peripheral adrenergic receptors. These clinical manifestations and complications are similar of those resulting from cocaine use and may be indistinguishable except for the duration of effect of amphetamines, which tends to be longer (up to 24 hours). Tachycardia and hypertension are the most common manifestations of cardiovascular toxicity. Most patients present to the emergency department, however, because of the central nervous system manifestations, these patients are anxious, volatile, and aggressive and may have life-threatening

agitation. Visual and tactile hallucinations and psychosis are common. Other sympathetic findings include mydriasis, diaphoresis, and hyperthermia. Death from amphetamine intoxication most commonly results from dysrhythmias, hyperthermia and intracerebral hemorrhage. Direct central nervous system toxicity may result in seizures. Hypertension, and vasospasm may lead to cerebral infarction and intrasparenchymal and subarachnoid hemorrhages. Tachycardia and hypertension can cause myocardial ischemia or infarction and aortic dissection. Dysrhythmias vary from premature ventricular complexes to ventricular tachycardia and ventricular fibrillation. Agitation, increased muscular activity and hyperthermia can result in metabolic acidosis, rhabdomyolysis, acute tabular necrosis (acute renal failure) and coagulopathy."

Mr. Price exhibited nearly all of these conditions, with the exception of the intraparenchymal and subarachnoid hemorrhage. In essence, what we see in Mr. Price is an individual with amphetamine toxicity as the single greatest contributing factor to his death. There is simply no evidence to suggest that hogtying played any role in his death. We have clear data that there is no respiratory component to the hogtie position. We also have clear data that Mr. Price was a chronic methamphetamine abuser. He had essentially all of the signs and symptoms of methamphetamine use and he died a death that was completely consistent with toxic delirium secondary to methamphetamine use. To suppose that anything else played a significant role in his death is speculation.

(iv) DR. REAY'S TRIAL TESTIMONY DECLARATION

Dr. Reay makes statements in his trial declaration that cannot be reconciled with his prior statements, nor can they be reconciled with

established respiratory physiology. At paragraph 5, page 2, line 1, of his declaration he says that "The primary focus of my study was not the measurement of oxygen levels following this exertion, but rather the impact on a restrained person's ability to breathe...." Yet, in his original study, he says, "These results show that positional restraint can prolong recovery from exercise as determined by changes in peripheral oxygen saturation and heart rate." He makes no mention in that study whatsoever about any measurements of an individual's ability to breathe. In his paper he never mentions ability to breathe, rather he writes about prolonged recovery from exercise. But once again, the abnormality he reported with exercise, is an abnormality which doesn't exist. You cannot have a prolonged recovery from something that doesn't exist in the first place. What the UCSD study showed was that the lungs function in such a fashion that you cannot distinguish from somebody who is hogtied and somebody who is not hogtied, as far as any gas exchange parameter you choose to measure (whether oxygen, carbon dioxide, Ph or chemical blood changes that are associated with post exercise responses) is concerned. The post exercise response is exactly the same in the prone hogtie position and a non-hogtie position.

At paragraph 5, page 2, line 24 of his declaration, he says, "The fact remains that regardless of whether oxygen levels increase following exertion that condition presupposes that the lungs are able to replenish the oxygen in a person's system." This makes no sense. First, PO_2 increases with exertion. Since blood oxygen levels don't go down, you don't have to replenish oxygen levels in the blood. That Dr. Reay talks about replenishing blood oxygen levels as a result of exercise demonstrates he has important misconceptions about

respiratory and exercise physiology. Obviously, there is a recovery from exercise. Everybody knows that. But the recovery is precisely the same as far as carbon dioxide levels, oxygen levels, and Ph levels are concerned, in the hogtie and non-hogtie position.

Dr. Reay comments why you gasp for air after exercise. On page 2, line 27, he says, "That is why a person who is winded after a long sprint run, is often times gasping for air, because that person has increased oxygen needs." However, that's not why you are winded after a long sprint. You are winded because you have developed a metabolic (lactic) acidosis. Blood Ph goes down with heavy exercise. Your body responds to try and maintain the Ph in a more normal range, so you breathe fast to lower the carbon dioxide level in the blood. Carbon dioxide is an acid, so if you have less acid, your Ph comes up compensating for the metabolic acidosis.

At paragraph 13, line 7, of Dr. Reay's trial testimony declaration, after acknowledging that the hogtie is "inherently neutral" he offers the idea that its use could be fatal "*if* there is then some weight placed upon the back of the individual [who is hogtied] and that individual is unable to move his chest." Dr. Reay's opinion regarding the death in this case depends on two suppositions: one, application of weight on a person's back of a sufficient amount and two, it keeps a person from moving his chest. I see no evidence for that in the depositions, the witnesses, or anywhere else in the records with which I was supplied. There were two witnesses who indicated that one of the deputies had a knee in the small of Mr. Price's back, but those witnesses indicated that occurred while they were trying to handcuff him. No weight was put on Price's back and no one sat on his back after he was hogtied. Although I can't say what

affect 270 pounds on somebody's back would be as far as the respiratory function is concerned (because I didn't measure it), I see no evidence that that occurred in this case.

As concerns, a knee in Price's back, whether after the hogtie or before the hogtie, if having a knee in your back caused asphyxia after struggle, along with being overweight, and being on your stomach, I think we can safely say that there would not be a single professional wrestler alive today.

Looking at paragraph 9, page 4, line 24 of Dr. Reay's declaration, one of the criticisms leveled at our study by him is that in our experiment we did not load subjects with methamphetamine. The criticism is preposterous. There is no way a Human Use Committee in the United States would permit an experiment to figure out whether or not exercise combined with methamphetamine might cause death due to cardiac arrest. You might be able to do it in animals, but even in animals, it might not be allowed because it is so obvious that methamphetamine kills people.

More importantly, our study was meant to examine whether the hogtie position could cause asphyxia, not whether exercise and amphetamines could cause death.

Another criticism Dr. Reay levels at our study and its applicability to this case is that none of the subjects were pressed on 134 degree asphalt which creates stress factors in people. It's true we did not put our subjects on hot asphalt, however, does Dr. Reay's observation imply that the deputies should have restrained Mr. Price in the air, rather than on the ground? But again more importantly, we examined whether the hogtie causes asphyxia, and it does not.

At paragraph 11, page 5, line 19 of his declaration, Dr. Reay states, "Dr. Neuman and the UCSD study did not refute or negate the basic concept of positional asphyxia." We absolutely did. Our study positively shows that you don't get positional asphyxia from the hogtie position.

(v) DR. REAY'S DEPOSITION

Referring to pages 16 through 19 of Dr. Reay's deposition in this case, he says over and over that oxygen levels decrease with exercise and that the hogtie position prevents them from recovering. He says, "You are in a deficit that you can't recover from. You can't get your breath and somehow you are unable to get full expansion, take some deep breaths to reoxygenate your system, then you are in a deficit that you can't recover from," referring to the hogtie position. Dr. Reay is wrong that you have to reoxygenate your blood. You simply don't desaturate with exercise. I believe Dr. Reay may have acknowledged he is wrong. For example, in his April 1997 deposition in the case of *Estate of Brinks v. City of Bonny Lake*, he testified at page 18, "an individual who has also exercised can tolerate the face-down hog-tied position without any physiological significance." He also concedes the hogtie position is "inherently neutral" in this case in his trial declaration (as noted previously).

At page 21 of his deposition in this case, he says a saturation level of 60 to 70% corresponds to hypoxia. Earlier I explained the consequences of an oxygen saturation of 85%. A 60 to 70% level would render most people unconscious. In fact, the blood that returns to the lungs to get reoxygenated after going through the body, is usually 75% saturated. 60 to 70% is profoundly hypoxic. Most people would be unconscious and probably would die in short order with oxygen

saturations that low because an oxygen saturation of 60% represents a PO_2 of about 30 mmHg. That's a number that would be rapidly fatal. Finally Dr. Reay acknowledges the fallacy of his hogtie hypothesis when he says at page 54, line 15 of his deposition in this case that, "If exercise increases oxygen saturation then the hogtie couldn't cause positional asphyxia."

(vi) CPR

Regarding CPR, the plaintiffs have submitted the proposition that Price would have survived had the deputies given him CPR. There is no medical evidence to support that. People with toxic delirium are most frequently not resuscitated. The notion that if you have a cardiac arrest somebody is going to come by and do CPR on you, they're going to take you to the hospital, and that everything is going to be fine after that, is a tremendous misconception on the part of the American public. Neurologically intact survival from cardiac arrest when CPR is given properly and promptly is in the neighborhood of a couple a percent. So, you can never say that somebody would have been resuscitated successfully because the odds of the successful resuscitation are so small to start with. I say this based not only on the literature, but based on my experience as an emergency room physician.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct to the best of my knowledge.

DATED: 11/3/97

TOM NEUMAN, M.D.